J. Parasitol., 94(6), 2008, pp. 1289–1294

FATAL INTESTINAL COCCIDIOSIS IN A THREE-WEEK-OLD BUFFALO CALF (*BUBALUS BUBALUS*)

J. P. Dubey, W. Wouda*, and J. Muskens*

Animal Parasitic Diseases Laboratory, Animal and Natural Resources Institute, Beltsville Agricultural Research Center, United States Department of Agriculture, Beltsville, Maryland 20705-2350. *e-mail: jitender.dubey@ars.usda.gov*

ABSTRACT: The water buffalo (*Bubalus bubalus*) is important to the economy of several countries, especially in Asia and South America. Little is known regarding the impact of coccidiosis in buffaloes. Cattle and buffaloes are considered to have common species of *Eimeria*, but critical cross-transmissions have not been made because it is difficult to raise these hosts coccidian free. Clinical coccidiosis was confirmed post mortem in a 22-day-old buffalo calf that died after a 3- to 4-day illness. Oocysts morphologically identical to *Eimeria bareillyi* were found in the feces and in sections of small intestine. Oocysts were often pyriform, sometimes with asymmetrical sides. The shorter end was flattened and approximately 5–6 μm wide. Unsporulated oocysts in feces were 23.2–29.5 × 16.5–22 μm, with an average of 27.2 × 19.3 μm. Schizonts, gamonts, and oocysts were identified in sections of small intestine where they were located in enterocytes of the jejunum and ileum. No coccidian stages were seen in sections of colon. This is one of the first confirmed cases of clinical coccidiosis in water buffalo.

At least 12 species of *Eimeria* parasitize cattle (*Bos taurus*) (Levine and Ivens, 1967; Levine, 1973). Of these, 5 species, *E. bovis*, *E zuernii*, *E. auburnensis*, *E. ellipsoidalis*, and *E. alabamensis*, are considered pathogenic. Clinical coccidiosis due to *E. zuernei* and *E. bovis* results in huge losses to cattle industry worldwide (Fitzerald, 1980). In general, eimerians are site and host specific. For example, eimerians of sheep and goats are morphologically identical, but they are considered separate species based on transmission experiments (Levine, 1973).

The water buffalo (*Bubalus bubalus*) is important to the economy of several countries, especially in Asia and South America. It is the main dairy animal species in India. Little is known regarding the impact of coccidiosis in buffaloes. Cattle and buffaloes are considered to have common species of *Eimeria* (Levine, 1973), but critical cross-transmissions have not been made because it is difficult to raise these hosts coccidian free. Additionally, *E. bareillyi* has been described in water buffalo (Gill et al., 1963). In a study from India, *E. bareillyi* was nontransmissible to cattle, whereas *E. zuernii* and *E. ellipsoidalis* were cross-transmissible (Sanyal et al., 1985). In addition to several reports of *E. bareillyi* infection in buffaloes in India, coccidian oocysts have been found in buffaloes from Italy, Turkey, and Brazil. Restani and Tassi (1969) reported *E. bareillyi* oocysts in feces of 23 of 162 buffalo calves from 20 farms in Italian provinces of Caserta and Latina; no mention was made of clinical signs associated with oocysts. Riebeiro et al. (2000) reported eimerian oocysts in all 106 young Murrah buffalo calves from Ribeira Valley, São Paulo state, Brazil; 48 of these calves had diarrhea. Sayin (1968) described oocysts of 11 species in 93.5% of 130 apparently healthy buffaloes from 5 provinces of Turkey. Three, 1-wk-old cow calves fed a mixture of *E. bovis*, *E. zuernii*, *E. ellipsodalis*, *E. auburnensis*, and a new species, *E. ankarensis* from buffaloes, shed oocysts of *E. bovis*, *E. zuernii*, *E. ellipsodalis*, and *E. auburnensis* but not *E. ankarensis*. *Eimeria bovis*, *E. suspherica*, and *E. clyinderica* oocysts were reported in feces of 20 Murrah buffalo calves from Malaysia (Sani and Chandrawathani, 1987). Here, we report fatal coccidiosis for a buffalo calf from The Netherlands.

MATERIALS AND METHODS

A water buffalo farm was started in 2000 in the province of Limburg in The Netherlands by the purchase of 70 adult buffaloes imported from southern Italy. Since that time, only bulls originating from other Dutch buffalo farms were added to the herd. During November and December 2007, 22 calves were born on this farm, 9 of which died after a period of diarrhea. The calves were born on a slatted floor and then separated from the mother. Most of the calves were housed individually in an igloo for 1 mo and subsequently housed together with other calves. The calves received colostrum from their own dam during the first 2 days after birth and after that they were fed milk replacer. The clinical signs of the affected calves included watery diarrhea (without blood), emaciation, and dullness. The calves kept drinking milk until shortly before death. Treatment with an antibiotic was not successful.

Clinical signs of diarrhea were first seen in 6-wk-old calves that died after 3–4 days of illness. Later, diarrhea was seen in calves 3 wk of age and in even younger calves. These calves died 1–2 days after the farmer had recognized diarrhea. A post-mortem examination was performed on 1 calf that had died at an age of 22 days.

A bacteriological culture on blood agar was done from the small intestinal contents. Fecal sample from the colon was used for testing by commercial lateral flow immunoassays for *Cryptosporidium parvum*, bovine rotavirus, and bovine enteric coronavirus. The McMaster flotation method was used for isolating coccidial oocysts in a fecal sample from the colon. Tissue samples of jejunum, ileum, and colon were placed overnight in 10% buffered formalin and routinely processed for paraffin embedment. Sections were cut at 4 μm and stained with hematoxylin and eosin and the periodic acid Schiff reaction. After examination, additional sections were cut at 1 μm to obtain better detail of parasitic stages. Small portions of formalin fixed small intestine were processed for transmission electron microscopy.

RESULTS

The carcass was emaciated. Thoracic organs were unremarkable except for some minor fibrous pleural adhesions. Abdominal organs were grossly normal except for the intestinal tract. There was some watery fluid and milk in the rumen and curdled milk in the abomasum. The small intestines were severely congested and edematous, and a loop of the lower small intestine seemed to be twisted and loosely attached to surrounding intestinal loops by delicate fibrin deposits. There was a small amount of watery contents in the small and the large intestines.

No enteropathogenic bacteria were found at culture from the

Received 23 April 2008; revised 1 May 2008; accepted 5 May 2008.
* GD-Animal Health Service, P.O. Box 9, 7400 AA Deventer, The Netherlands.

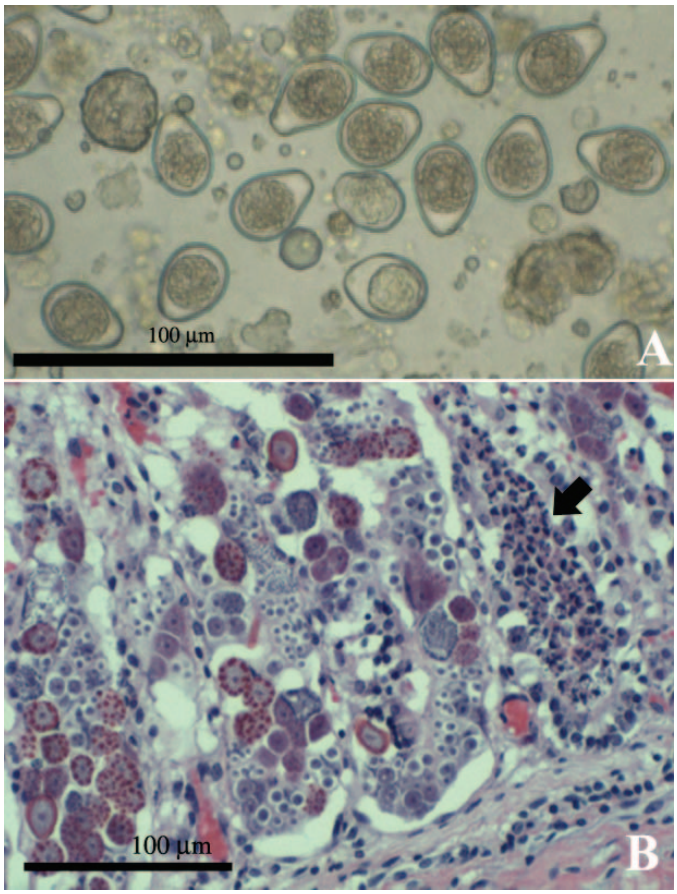

FIG. 1. (**A**) Fecal float from buffalo calf showing unsporulated *Eimeria bareilyi*-like pear-shaped oocysts. (**B**) Section of jejunum with heavy parasitization with gamonts and oocysts, and a crypt abscess (arrow).

small intestine. No enteropathogenic antigens were detected by lateral flow immunoassays for *C. parvum*, bovine rotavirus, or bovine enteric coronavirus. Large numbers of unsporulated oocysts were found in the fecal sample from the colon (Fig. 1A). All 71 oocysts in 2 microscopic fields were measured from the photographs. Oocysts were often pyriform, sometimes with asymmetrical sides. The shorter end was flattened and approximately 5–6 μm wide. Oocysts were 23.2–29.5 × 16.5–22 μm, with an average of 27.2 × 19.3 μm. The length–width ratio was 1.26–1.57, with an average of 1.38. Oocysts had 2 walls; the outer wall was missing from the flattened end (Fig. 1A).

Endogenous stages of a coccidian were seen in enterocytes in histological sections from the jejunum and ileum but not colon. Stages were seen throughout the villus, but the crypts of Lieberkühn were more severely parasitized. In some fields, most enterocytes were parasitized by gamonts and oocysts (Fig. 1B). There seemed to be 2 types of schizonts based on size and structure of merozoites (Fig. 2A, B). The larger schizonts were approximately 15–20 μm and contained 4–16 merozoites (Fig. 3). The merozoites were sickle shaped, with the nucleus at the broader end; they were approximately 8 μm long (Fig. 2A). The smaller schizonts contained merozoites that were approximately 4 μm long and had a centrally located nucleus (Fig. 2). All stages of gametogony from trophozoites to mature oocysts

TABLE I. Characteristics of 5 pathogenic species of *Eimeria* in cattle.

Species	Location in intestine			Oocysts	Prepatent period (days)	Reference
	Schizonts		Gamonts			
	First	Second				
E. bovis	Macroscopic, in lamina propria of SI	Small in size in cecum and colon	Cecum and colon	Ovoid, 26.7 × 20.2	19	Hammond (1964); Hammond et al. (1946, 1966)
E. zuernii	Large, in lamina propria of SI	Small in size, cecum, colon	Cecum and colon	Spherical-ellipsoidal, 19 × 17.8	16–20	Stockdale (1977a, 1977b)
E. ellipsoidalis	Small in size, SI	Unknown	SI	Ellipsoidal, 23.1 × 16.1	9–12	Hammond, Sayin, and Miner (1963)
E. alabamensis	Small in size, SI	Unknown	SI, IN-	Ovoid, 20.7 × 14.8	6–8	Davis et al. (1957)
E. auburnensis	Large in size, SI	Small in size, SI	SI, microgamonts macroscopic	Elongate ovoid, 41.3 × 24.7	18	Chobotar and Hammond (1969); Chobotar et al. (1969)

SI = small intestine; IN = intranuclear.

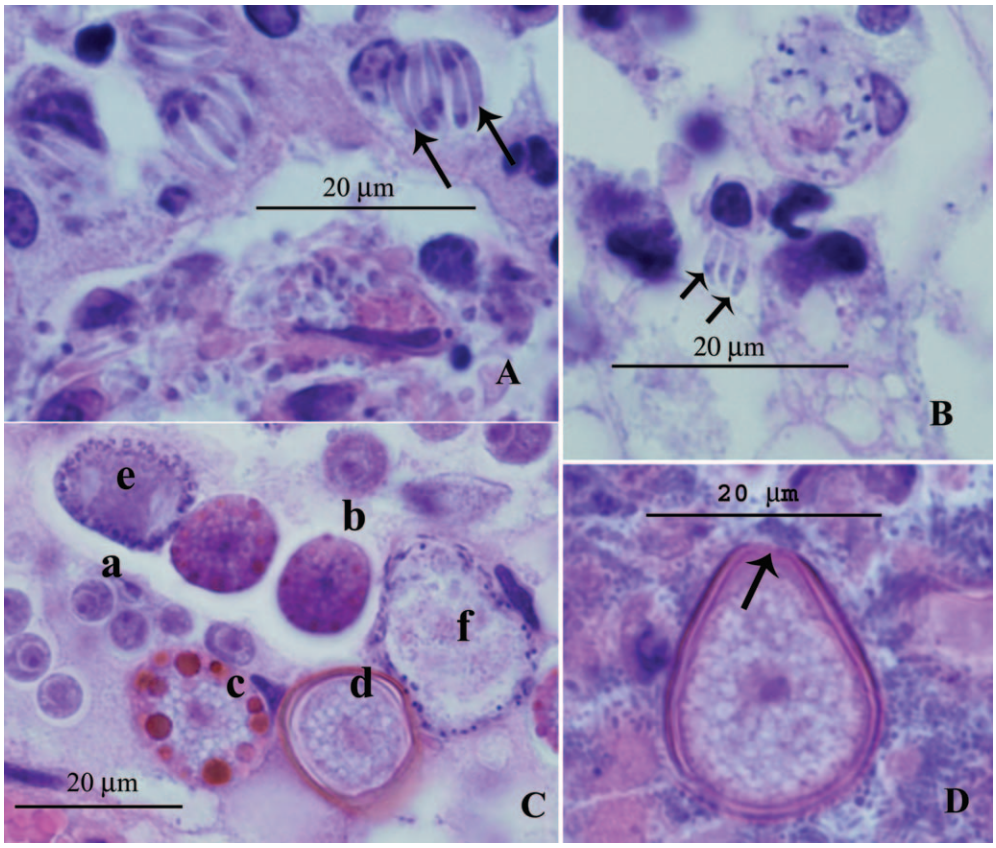

FIG. 2. Endogenous stages of *Eimeria bareillyi* in sections of ileum of the calf. (**A**) Schizonts in enterocytes. The crypt lumen is plugged with degenerating merozoites. Note elongated merozoites with terminal nucleus (arrows). (**B**) A small schizont with 3 small merozoites (arrows). Compare the sizes of merozoites in A and B; both photos are of comparable sizes. A microgamont is located above the schizont. (**C**) Gametogonic stages. (a) Small macrogamont with a large nucleus, (b) nearly mature macrogamont with a central nucleus, (c) macrogamont with eosinophilic wall-forming bodies, (d) an oocyst, (e) immature microgamont with peripherally located nuclei, and (f) microgamont with peripheral microgames. (**D**) A longitudinally cut oocyst with 2 oocyst walls and a micropyle (arrow).

were seen, often in adjacent cells (Fig. 2). The microgamonts were up to 20 µm and contained numerous microgamtes (Fig. 2C), sometimes around an eosinophilic residual body. Oocysts were unsporulated and resemembled those seen in the feces (Fig. 2D). Two longitudinally cut oocysts were 23 × 18.5 and 23.5 × 15.6 µm. There was destruction of crypt enterocytes and accumulation of desintegrating polymorphonuclear leucocytes within crypts (crypt abscesses; Figs. 1B, 2A). Ileal Peyer's patches showed lymph depletion.

By transmission electron microscopy (TEM), immature and mature schizonts, merozoites, gamonts, and oocysts were found in enterocytes (Fig. 3A, B). Longitudinally cut merozoites were 7.0–8.5 × 1.3–1.5 µm (n = 7) (Fig. 3B). The position of the nucleus in longitudinally cut merozoites varied from being subterminal to terminal (Fig. 3). Although merozoites were autolyzed, numerous micronemes could be recognized (Fig. 3B). Merozoites were often located with their conoidal and nonconoidal ends in opposite directions within a schizont (Fig. 2A). Immature schizonts contained nuclei that were located centrally and could be distinguished from immature microgamonts that had peripherally located nuclei and a central residual body. Macrogamonts contained wall-forming bodies and amylopectin.

DISCUSSION

Morphology of oocysts and endogenous stages, and their location in the host, are helpful in differential diagnosis of eimerian species (Table I). Eimerians of cattle undergo asexual schizogony once or twice before the gametogonic development. In this respect, both *E. zuernii* and *E. bovis* are unusual. First generation schizonts of both of these species are large and even macroscopic, and they occur in the lamina propria of small intestine. *Eimeria bovis* schizonts parasitize endothelial cells of lacteals (Hammond, 1964). *Eimeria zuernii* first generation schizonts also develop in unknown cells of the lamina propria (Stockdale, 1976). Their second generation schizonts are found in enterocytes of the cecum and colon and are relatively small; in histological sections, *E. bovis* schizonts are approximately 10 µm wide, whereas those of *E. zuernii* are about 15 µm wide (Hammond, Andersen, and Miner, 1963; Stockdale, 1976). Gamonts of both species occur in cecum and colon and are considered the cause of diarrhea and dysentery. From a diagnostic point of view, schizonts of *E. zuernii* are more numerous and occur simultaneously with gamonts (Smith and Graybill, 1918; Stockdale, 1977a, 1977b), whereas those of *E. bovis* are few in number and generally not together with gamonts. The

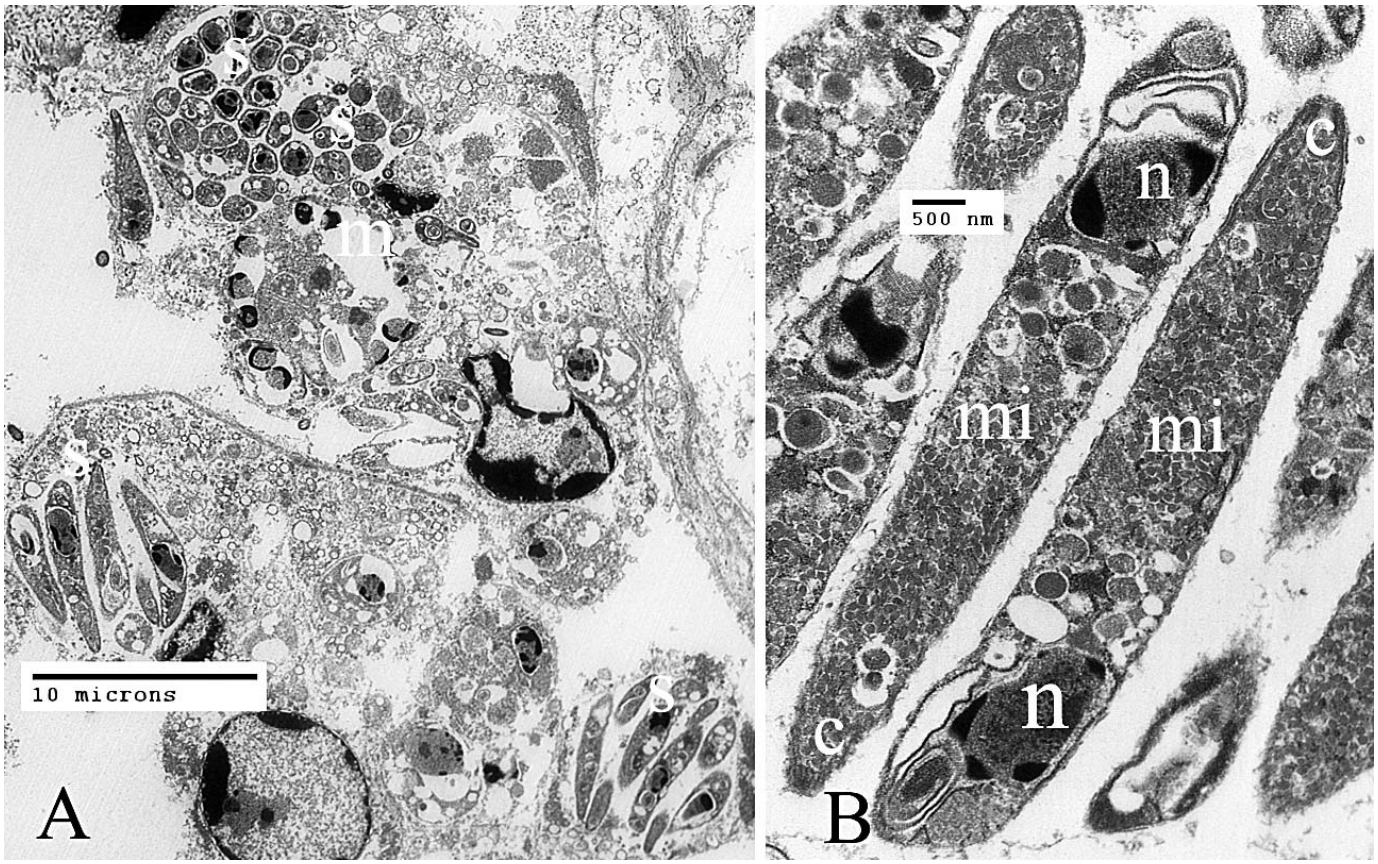

FIG. 3. TEMs of *Eimeria bareillyi* in sections of jejunum. (**A**) Four schizonts (s), a microgamont and several individual merozoites. (**B**) Two longitudinally cut merozoites . Note conoid (c), numerous micronemes (mi), and terminal nucleus (n).

prepatent period for *E. bovis* and *E. zuernii* is approximately 19 days, and clinical signs coincide with shedding of oocysts. Clinical coccidiosis frequently occurs in calves 1–6 mo of age, but older animals are also affected. *Eimeria zuernii* seems to cause disease more frequently in older cattle than *E. bovis* (Hammomd, 1964).

The biology of *E. ellipsoidalis* differs from *E. bovis* and *E. zuernii* because it develops in the small intestine, endogenous stages are short lived, and the prepatent period is 10 days; the diarrhea produced, if any, is not bloody. Schizonts are small (10.6 × 9.4 μm wide) and contain 24–36 merozoites; the number of generations of schizonts is unknown (Hammond, Sayin, and Miner, 1963)

The life cycle of *E. alabamensis* is unusual because all endogenous stages occur in the nucleus of small intestinal enterocytes (Davis et al., 1955, 1957; Svensson, 1994). The prepatent period is 18 days. Clinical disease occurs in older grazing calves (Svensson et al., 1994).

Eimeria auburnensis occurs in the small intestine. Its schizonts are deeply embedded and gamonts are found in mesodermal cells; and microgamonts can be macroscopic (Chobotar and Hammond, 1969; Chobotar et al., 1969). The prepatent period is 18 days.

Based on these findings, the parasite in the present study is not ascribed to any of the species discussed in Table I. However, it most closely resembles *E. bareillyi*, first described from buffaloes in India (Gill et al., 1963).

Eimeria bareillyi oocysts are morphologically distinct than other species of *Eimeria* (Gill et al., 1963; Bhatia et al., 1968). The oocysts are pear shaped, with a flattened anterior end. In experimentally infected calves, the minimum prepatent period was 14 days (Shastri and Ghafoor, 1982; Sanyal et al., 1984/1985a, 1984/1985b). Its endogenous development was studied in 3, 2-day-old buffalo calves killed on days 5, 10, and 14 after feeding 1 million oocysts (Shastri and Ghafoor, 1982). Development occurred in the jejunum and ileum. On day 5 postinoculation (PI), only uninuclear parasites (trophozoites) were found in mid-jejunum. On day 10 PI, schizonts were found in enterocytes of the crypts of Lieberkühn. Mature schizonts were 16.2 (11.5–20.7) μm in diameter and contained 24–36 merozoites. The merozoites were 11.7 × 1.9 (10–15 × 1.5–2.5) μm; whether these measurements were made from smears or sections was not stated. On day 14 PI, only gamonts were found. Macrogamonts were 19.2 × 11.7 (12.5–23.3 × 11.1–16.7), microgamonts were 15–30 × 10–23 μm, and oocysts in sections were 25.3 × 16.9 (21.7–31.7 × 15–20 μm. Coccidian stages were not found in the fourth control calf killed day 15 PI (Shastri and Ghafoor, 1982). Before this study, Pande, Bhatia, and Chauhan (1971) reported gamonts and oocysts of *E. bareillyi* in the jejunum of 2 naturally infected buffalo calves of un-

known age and unknown clinical status. Lesions associated with these stages were macroscopic and characterized by 3–6 mm whitish opalescent polyplike areas (Pande, Bhatia, and Chauhan, 1971). Only gamonts and oocysts (no schizonts) were found by examination of sections and scrapings of the macroscopic lesions.

In the present study, the longest longitudinally cut merozoites measured using TEM photographs were 7–8.5 μm long and thus were smaller than that stated by Shastri and Ghafoor (1982). However, the latter authors did not state whether the measurements were made on merozoites in smears or in sections.

Under certain circumstances, *E. bareillyi* can be pathogenic. Shastri et al. (1974) found clinical coccidiosis associated with *E. bareillyi* in 16.7% of 90 buffalo calves born during 1969–1971 in an Indian dairy farm. Most severe disease was in 18- to 45-day-old calves, and clinical signs began 1–2 day before shedding of oocysts. The affected calves were anorexic and had yellowish white mucoid watery feces. Untreated calves became moribund 4–7 days after the onset of illness. Ten of these calves were examined post mortem (Shastri and Krishnamurthi, 1975). Whitish opalescent 2–6 mm wide areas were found in jejunum and ileum; numerous gamonts and oocysts of *E. bareillyi* were present in scrapings made from these macroscopic lesions (Shastri and Krishnamurthi, 1975). Similar macroscopic lesions were reported by Shastri et al. (1976) in 7 naturally infected buffalo calves that had mixed *E. bareillyi* and *E. ellipsoidalis* infections.

Experimentally, *E. bareillyi* was found to be pathogenic (Shastri et al., 1973; Sanyal et al., 1984/1985a,1984/1985b). Calves fed 50,000 or more oocysts became sick (Shastri et al., 1973). In an anticoccidial drug trial, 20, 1-mo-old buffalo calves were each dosed with 100,000 sporulated oocysts of *E. bareillyi* and divided in to 5 groups of 4 each (Sanyal et al., 1984/1985b). All 20 calves shed *E. bareillyi* oocysts. All 4 untreated calves developed diarrhea of 5- to 8-day duration and 2 died of coccidiosis on day 18 PI. The groups of calves medicated orally with sulfadiamidine (30 mg/kg body weight) for 4 days 10–13 days PI or with amprolium (10 mg/kg) orally for 10–19 days PI remained asymptomatic. Two groups of calves medicated with chloroquine (500 mg, once for 10–13 days PI) or halofuginone (1.2 mg/kg, once on day 10 PI) were ineffective in preventing clinical signs (Sanyal et al., 1984/1985b).

We are not aware of reports of clinical coccidiosis associated with *E. bovis*, *E. zuernii*, or other eimerian species in buffaloes. Endogenous stages considered to be of *E. bovis*, *E. ellipsoidalis*, *E. auburnensis*, and other eimerians were reported in histological sections of intestines from buffalo calves from the slaughterhouse (Patnaik and Pande, 1965; Bhatia and Pande, 1967; Pande, Bhatia, Chauhan, and Garg, 1971; Shastri et al., 1976); clinical status was unknown.

ACKNOWLEDGMENTS

We thank Han Gussinklo and Coen Hegeman for parasitological examination of fecal samples; John Jenkins, Armed Forces Institute of Pathology, Washington, D.C., for electron microscopy; and G. V. Velmurugan for help in preparation of photo plates.

LITERATURE CITED

BHATIA, B. B., AND B. P. PANDE. 1967. Giant eimerian schizonts in the Indian water-buffalo. Acta Veterinaria Academiae Scientiarum Hungaricae **17:** 351–357.

———, ———, P. P. S. CHAUHAN, AND G. S. ARORA. 1968. A study on the sporulated oocysts of twelve eimerian species in Indian buffalo (*Bulbalus bubalis*). Acta Veterinaria Academiae Scientiarum Hungaricae **18:** 115–133.

CHOBOTAR, B., AND D. M. HAMMOND. 1969. Development of gametocytes and second asexual generation stages of *Eimeria auburnensis* in calves. Journal of Parasitology **55:** 1218–1228.

———, ———, AND M. L. MINER. 1969. Development of the first-generation schizonts of *Eimeria auburnensis*. Journal of Parasitology **55:** 385–397.

DAVIS, L. R., D. C. BOUGHTON, AND G. W. BOWMAN. 1955. Biology and pathogenicity of *Eimeria alabamensis* Christensen, 1941, an intranuclear coccidium. American Journal of Veterinary Research **59:** 274–281.

———, ———, AND ———. 1957. The endogenous development of *Eimeria alabamensis* Christensen, 1941, an intranuclear coccidium of cattle. Journal of Protozoology **4:** 219–225.

FITZGERALD, P. R. 1980. The economic impact of coccidiosis in domestic animals. Advances in Veterinary Science and Comparative Medicine **24:** 121–143.

GILL, B. S., M. B. CHHABRA, AND N. B. LALL. 1963. A new species of coccidium, *Eimeria bareillyi* n. sp. from buffaloes. Archives für Protistenkunde **106:** 571–574.

HAMMOND, D. M. 1964. Coccidiosis of cattle. Some unsolved problems. *In* Thirtieth Faculty Honor Lecture, the Faculty Association. Utah State University, Logan Utah, p. 1–38.

———, F. L. ANDERSEN, AND M. L. MINER. 1963. The occurrence of a second asexual generation in the life cycle of *Eimeria bovis* in calves. Journal of Parasitology **49:** 428–434.

———, G. W. BOWMAN, L. R. DAVIS, AND B. T. SIMMS. 1946. The endogenous phase of the life cycle of *Eimeria bovis*. Journal of Parasitology **32:** 409–427.

———, J. V. ERNST, AND M. L. MINER. 1966. The development of first generation schizonts of *Eimeria bovis*. Journal of Protozoology **13:** 559–564.

———, F. SAYIN, AND M. L. MINER. 1963. Über den Entwicklungszyklus und die Pathogenität von *Eimeria ellipsoidalis* Becker und Frye, 1929, in Kälbern. Berliner und Münchener Tierärztliche Wochenschrift **76:** 331–332.

LEVINE, N. D. 1973. Protozoan parasites of domestic animals and of man, 2nd ed. Burgess Publishing Co., Minneapolis, Minnesota, 406 p.

———, AND V. IVENS. 1967. The sporulated oocysts of *Eimeria illinoisensis* n. sp. and of other species of *Eimeria* of the ox. Journal of Protozoology **14:** 351–360.

PANDE, B. P., B. B. BHATIA, AND P. P. S. CHAUHAN. 1971. Sexual stages and associated lesion in *Eimeria bareillyi* of buffalo calves. Indian Journal of Animal Sciences **41:** 151–154.

———, ———, ———, AND R. K. GARG. 1971. Parasitic lesions in cow calves (below two years): A histologic study. Indian Journal of Animal Sciences **41:** 345–350.

PATNAIK, M. M., AND B. P. PANDE. 1965. Some observations on the endogenous stages of species of *Eimeria* and related lesions in naturally infected buffalo-calves (*Bubalus bubalis*). Indian Journal of Veterinary Science **35:** 33–46.

RESTANI, R., AND P. TASSI. 1969. *Eimeria bareillyi* in bufali Italiani. Atti della Societa Italiana delle Scienze Veterinarie **23:** 881–885.

RIBEIRO, M. G., H. LANGONI, J. A. JEREZ, D. S. LEITE, F. FERREIRA, AND S. M. GENNARI. 2000. Identification of enteropathogens from buffalo calves with and without diarrhoea in the Ribeira Valley, State of São Paulo, Brazil. Brazilian Journal of Veterinary Research and Animal Science **37:** 159–165.

SANI, R. A., AND P. CHANDRAWATHANI. 1987. Coccidia of buffalo calves. Tropical Biomedicine **4:** 190–191.

SANYAL, P. K., N. S. RUPRAH, AND M. B. CHHABRA. 1984/1985a. Evidence of cell mediated immune response in infection with *Eimeria bareillyi* in buffaloes. Veterinary Parasitology **17:** 111–115.

———, ———, AND ———. 1984/1985b. Chemotherapeutic efficacy of sulphadimidine, amprolium, halofuginone and chloroquine phosphate in experimental *Eimeria bareillyi* coccidiosis of buffaloes. Veterinary Parasitology **17:** 117–122.

———, ———, AND ———. 1985. Attempted transmission of three species of *Eimeria* Schneider, 1875 of buffalo-calves to cow-calves. Indian Journal of Animal Sciences **55:** 301–304.

SAYIN, F. 1968. The sporulated oocysts of *Eimeria ankarensis* n. sp. and of other species of *Eimeria* of buffalo in Turkey and transmission of four species of *Eimeria* from buffalo to cow-calves. Ankara Universitesi Veteriner Fakultesi Dergisi **15:** 282–300.

SHASTRI, U. V., AND M. A. GHAFOOR. 1982. Observations on the developmental stages of *Eimeria bareillyi* Gill, Chhabra and Lall, 1963, in buffalo-calves. Indian Journal of Animal Sciences **52:** 309–313.

———, AND R. KRISHNAMURTHI. 1975. A note on pathological lesions in clinical bubaline coccidiosis due to *Eimeria bareillyi*. Indian Journal of Animal Sciences **45:** 46–47.

———, ———, AND M. A. GHAFOOR. 1973. Some observations on pathogenicity of *Eimeria bareillyi* in buffalo calves. Indian Journal of Animal Research **7:** 23–25.

———, ———, AND ———. 1974. A note on clinical coccidiosis in buffalo calves due to *Eimeria bariellyi* Gill, Chhabra and Lall, 1963 in Maharashtra. Indian Veterinary Journal **51:** 301–302.

———, M. A. GHAFOOR, AND R. KRISHNAMURTHY. 1976. Some observations on coccidian lesions associated with endogenous phases in buffalo calves. Indian Journal of Animal Sciences **46:** 556–561.

SMITH, T., AND H. W. GRAYBILL. 1918. Coccidiosis in young calves. Journal of Experimental Medicine **28:** 89–108.

STOCKDALE, P. H. G. 1976. Schizogony and gametogony of *Eimeria zuernii* (Rivolta, 1878) Martin, 1909. Veterinary Parasitology **1:** 367–376.

———. 1977a. Proposed life cycle of *Eimeria zuernii*. British Veterinary Journal **133:** 471–473.

———. 1977b. The pathogenesis of the lesions produced by *Eimeria zuernii* in calves. Canadian Journal of Comparative Medicine **41:** 338–344.

SVENSSON, C. 1994. Bovine coccidiosis with special reference to *Eimeria alabamensis* infections in grazing calves. Ph.D. Thesis. Swedish University of Agricultural Sciences, Skara, Sweden, 48 p.

———, A. UGGLA, AND B. PEHRSON. 1994. *Eimeria alabamensis* infection as a cause of diarrhea in calves at pasture. Veterinary Parasitology **53:** 33–43.